Commentary

Malignant superior vena cava syndrome

The authors have described a case of malignant superior vena cava (SVC) syndrome secondary to lung metastases of urothelial carcinoma.

SVC syndrome was first described by the Scottish physician, William Hunter, in 1757 in a patient who died of aortic aneurysm.[1] The constellation of clinical features in SVC syndrome occurs as a result of obstruction, either intrinsic or extrinsic, of SVC. The classical clinical features include facial, periorbital, neck and arm swelling, and dilated superficial veins over the chest wall due to increased venous pressure in the upper body. Other more distressing complications include cough, hoarseness, dyspnea, stridor, and dysphagia due to compromise of the larynx or pharynx by the edema. Cognitive dysfunction may also occur due to cerebral venous hypertension.[2] In today's time, thoracic malignancy and thrombosis (secondary to increased use of intravascular devices such as catheters and pacemakers) constitute majority of the cases. The common malignant causes are non–small-cell lung cancer and small-cell lung cancer in approximately 50% and 25% of the patients, respectively. Metastatic cancers contribute to around 10% cases of SVC syndrome.[3]

Diagnosis of SVC syndrome is clinical, which is confirmed by radiological studies. Contrast-enhanced computerized tomography (CECT) scan is usually sufficient to arrive to a diagnosis of SVC syndrome.[3] Magnetic resonance imaging may be undertaken if the patient has allergy to contrast agent. Venogram is usually reserved for patients in whom surgery or endovascular stenting is contemplated. A tissue diagnosis is necessary to confirm the presence of malignant etiology. A thorough clinical examination may prove vital in determining if a peripheral biopsy site (e.g., a palpable supraclavicular lymph node) is accessible before embarking to an invasive procedure for tissue diagnosis. At times, simple sputum cytological examination or diagnostic thoracocentesis (if pleural effusion is present) may prove diagnostic and avoid more invasive tests (e.g., image-guided biopsy, bronchoscopy, or mediastinoscopy).

The management of SVC syndrome rests on the severity of symptoms, the cause of the obstruction, and the histological type of the tumor. Supportive treatment such as head elevation, rest, cautious administration of fluids, and supplemental oxygen is important even before a tissue diagnosis is obtained. Though the role of glucocorticoids is not yet proven in SVC syndrome, they are also prescribed for the symptomatic relief. Conventional definitive treatment modalities of SVC syndrome include irradiation, chemotherapy, or both, depending upon the histological type of tumor. Role of surgery is limited. Initial treatment success using radiotherapy or chemotherapy is reasonable and patient starts experiencing symptomatic relief within a week. Endovascular stenting is being increasingly used to manage SVC syndrome in the last 15 years. Initially, most of the authors reported endovascular stenting as a coadjuvant treatment if there had been little or no response to radiotherapy or chemotherapy or if the clinical syndrome recurred after conventional treatment. However, endovascular stenting is being promoted recently as the first-line therapeutic measure in all patients with SVC syndrome because stenting provides immediate and spectacular relief of symptoms (within 24-72 h after stent placement). Moreover, it does not interfere with subsequent antitumor treatments.[4-6] Furthermore, use of chemotherapy, radiotherapy, or both as the first-choice treatment of symptom relief in SVC syndrome is associated with a protracted waiting time of 3-4 weeks that eliminates early assessment of treatment effectiveness.

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